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Stroke

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## RESEARCH ARTICLE

# Kinematic Alterations in the Ipsilateral Shoulder of Patients with Hemiplegia Due to Stroke

## ABSTRACT

Meskers CGM, Koppe PA, Konijnenbelt H, Veeger HEJ, Janssen TWJ: Kinematic alterations in the ipsilateral shoulder of patients with hemiplegia due to stroke. *Am J Phys Med Rehabil* 2005;84:97–105.

**Objective:** To evaluate the assumption that shoulder kinematic patterns of the ipsilateral, nonparetic shoulder in hemiplegia are similar to kinematics recorded in a healthy population.

**Design:** Case control study of a convenience sample of ten patients with hemiplegia due to stroke in the subacute phase compared with a control group of similar age. Three-dimensional positions of the scapula and humerus were measured and expressed in Euler angles as a function of active arm elevation in the frontal and sagittal plane and during passive humeral internal/external rotation at an elevation angle of 90 degrees in the frontal and sagittal plane.

**Results:** Compared with controls, in the ipsilateral shoulder of patients, we found both a statistically significant diminished scapular protraction during elevation in the sagittal plane ( $35 \pm 5$  vs.  $51 \pm 8$  degrees at 110 degrees of humeral elevation) and humeral external rotation during arm elevation in the frontal plane ( $51 \pm 7$  vs.  $69 \pm 14$  degrees at 110 degrees of humeral elevation). Maximal passive humeral external rotation was found to be impaired in the frontal ( $64 \pm 13$  vs.  $98 \pm 14$  degrees) and sagittal planes ( $65 \pm 11$  vs.  $94 \pm 12$  degrees). In addition, there was significantly diminished anterior spinal tilt during humeral internal rotation ( $-5 \pm 10$  vs.  $-20 \pm 9$  degrees) and diminished posterior spinal tilt during external rotation in the frontal plane ( $-14 \pm 8$  vs.  $-3 \pm 6$  degrees). Maximal thoracohumeral elevation in patients was significantly impaired ( $126 \pm 12$  vs.  $138 \pm 8$  degrees).

**Conclusion:** Clear kinematic changes in the ipsilateral shoulder in patients with hemiplegia were found, indicating underlying alterations in muscle contraction patterns. The cause remains speculative. These results suggest that the ipsilateral shoulder should not be considered to function normally beforehand.

**Key Words:** Cerebrovascular Accident, Paresis, Shoulder Joint, Biomechanics

In stroke, functional impairment of the involved upper limb and the occurrence of shoulder pain are well known problems hampering the rehabilitation process. Remaining functional impairments of the upper limb are reported to vary from 21% to 67%.<sup>1,2</sup> The prevalence of shoulder pain as reported in the literature varies from 16% to 84%.<sup>3-9</sup> Pathogenesis is still unclear. Possible risk factors have been discussed thoroughly in the literature, and only a weak association has been found between shoulder pain and muscle tone,<sup>10</sup> although shoulder pain has also been found to be associated with adhesive capsulitis and disorders of the autonomic nervous system.<sup>10</sup>

Biomechanical analysis is necessary to gain more insight in the underlying mechanisms. A major advantage of such an analysis is that it addresses pathophysiologic mechanisms on an individual basis, thus bypassing the need to assess large groups of patients to find common denominators. Biomechanical analysis preferably starts with kinematic analysis because this is relatively easy to perform and serves as input for further biomechanical modeling using upper limb models such as the Swedish model<sup>11,12</sup> or the Delft Shoulder and Elbow Model.<sup>13-15</sup> Techniques to measure the kinematics of the shoulder, including the scapula with its large motion trajectory underneath the skin, were recently developed. Three-dimensional scapular positions can be measured in a reliable and fast way using a three-dimensional electromagnetic tracking device in combination with the palpation method and a scapula locator.<sup>16-21</sup>

Because of the relatively large interindividual variability,<sup>18,20</sup> it seems an obvious choice to compare kinematics of the paretic shoulder with the ipsilateral, nonparetic shoulder. Studying the literature, however, raised serious doubts concerning the assumption of normal kinematics in the ipsilateral shoulder, necessary for proper comparison. Several studies showed distinct changes in the con-

tralateral hemisphere in unilateral stroke in the form of significant magnetic resonance imaging changes,<sup>22</sup> a change in physiologic responsiveness after posterior temporal infarction,<sup>23</sup> and remote edema.<sup>24</sup> Others showed marked and lasting impairments in strength and coordination of the ipsilateral limb.<sup>1,25-28</sup> Hence, it may be that in the ipsilateral shoulder, scapular position disorders are present. The primary goal of this study was therefore to compare kinematics of the nonaffected ipsilateral shoulder of patients with hemiplegia due to stroke with a control group. Next to the main question of whether kinematics of the nonaffected side could be used as control in the study of shoulder kinematics in the hemiplegic shoulder, results of the study may have clinical consequences because kinematic alterations in the nonaffected shoulder with respect to a healthy population indicate altered muscle contraction patterns, resulting in improper function. Because patients with hemiplegia often have to rely on the ipsilateral side for activities of daily living, this may be important information for the clinician.

## METHODS

### Subjects

Ten patients, four men and six women (mean age  $\pm$  standard deviation, 53.4  $\pm$  10.3 yrs), with hemiplegia after stroke were recruited from the wards of the Rehabilitation Center Amsterdam (Table 1). None of the patients had a history of shoulder complaints; all had experienced their first stroke and were able to perform the measurements in physical, cognitive, and communicative sense. Before starting the measurements, shoulder pain, muscle tone, and arm function were objectified by a Visual Analog Scale, modified Ashworth<sup>29</sup> scale, and Fugl-Meyer<sup>30,31</sup> score, respectively. Information on neglect was obtained from the medical record.

Ten healthy subjects, six men and four women, of similar age (60.8  $\pm$  12.4 yrs) with a negative

**TABLE 1** Patients' characteristics. See text for further explanation

| Sex | Age, yrs | Paretic Side | Time Since Onset of Stroke, mos | Ashworth Scale Score | Fugl-Meyer Score | VAS Score | Neglect |
|-----|----------|--------------|---------------------------------|----------------------|------------------|-----------|---------|
| M   | 65       | R            | 10                              | 1                    | 6                | —         | N       |
| F   | 38       | R            | 7                               | 1                    | 3                | —         | N       |
| F   | 51       | R            | 1                               | 4                    | 4                | —         | N       |
| F   | 62       | L            | 11                              | 1                    | 2                | —         | N       |
| F   | 43       | L            | 15                              | 3                    | 2                | 7         | N       |
| F   | 46       | L            | 12                              | 3                    | 3                | 7         | Y       |
| M   | 49       | L            | 1                               | 5                    | 2                | 1         | N       |
| M   | 49       | L            | 6                               | 2                    | 2                | —         | N       |
| M   | 63       | L            | 7                               | 3                    | 2                | —         | N       |
| M   | 68       | L            | 7                               | 2                    | 2                | —         | N       |

VAS, Visual Analog Scale; M, male; F, female; R, right; L, left; N, no; Y, yes.

history of shoulder complaints formed the control group. For the lack of data on the differences in shoulder kinematics between men and women and because of the sample size, data of men and women were pooled. All control subjects took part in normal daily activities. Before the start of the measurements, the local board of medical ethics approved the study. Measurements were performed after each subject had signed an informed consent statement.

## Instrumentation

A MotionMonitor electromagnetic tracking device (Innovative Sports Training, Chicago, IL), consisting of a transmitter creating a weak magnetic field in which the position and orientation of several receivers can be measured, was used. A field calibration was performed after which the translational residual measurement error was about 2 mm for each coordinate and the rotational root-mean-square area was  $<2$  degrees for each axis of rotation.<sup>32</sup> Measurements were performed according to the protocol formulated by the International Shoulder Group Committee on standardized description of shoulder motion.<sup>33</sup> Receivers were attached to the thorax and upper arm by Velcro straps. One receiver was attached to a scapula locator: an adjustable tripod that is to be placed manually over the inferior angle, acromial angle, and scapular spinal triangle, respectively. A fourth receiver was attached on top of a pointer of about 2.5 cm in length, thus forming a spatial digitizer. Reliability, validity, and resolution of the measurement method were discussed previously.<sup>18</sup>

## Measurements

Subjects were seated in a plastic chair in front of the transmitter. Before starting the measurements, the scapula locator was adjusted to the scapula to be measured. First, a reference measurement was performed, during which a number of bony landmarks on the thorax, scapula, and humerus were digitized to be able to relate their position to the receivers attached to the bones. The three endpoints of the scapula locator were subsequently digitized after adjustment to relate their position to the attached receiver.

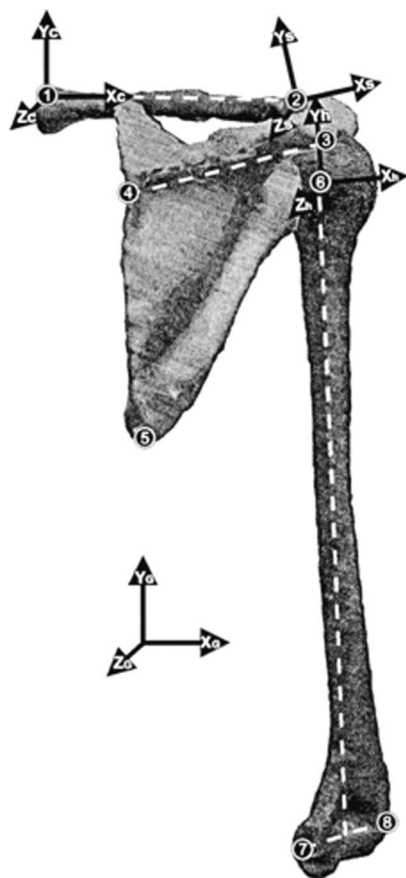
The positions of five bony landmarks (i.e., coracoid process, acromioclavicular joint, scapular acromial angle, spinal triangle, and inferior angle) were used to estimate the localization of the glenohumeral joint rotation center with respect to the receiver attached to the humerus.<sup>34</sup> The glenohumeral rotation center provides for the third essential landmark for the reconstruction of a local coordinate system on the humerus.

Both patients and control subjects performed

arm elevations in the frontal (forward flexion) and sagittal planes (abduction), respectively. The plane of elevation was dictated and controlled by means of a semicircular pipe with 10-degree marks, placed in the required elevation plane to guide the subject/patient. The subjects were asked to elevate their arm in steps dictated by the marks on the pipe. At each step, the subjects were asked to keep their arm still, allowing for the measurement of the scapular position to take place. Each elevation of the arm was therefore semistatic, subdivided in about 10–15 steps of about 10 degrees of elevation each. As the exact angle of humerus elevation was calculated afterward, the exact width of the elevation interval was not a critical variable. For each elevation angle, the scapula locator was repositioned over the scapula. Position and orientation of the receivers on the thorax, scapula locator, and humerus were subsequently recorded for each motion step. Subjects and patients were instructed to elevate their arm as high as possible, without further specification. Three subsequent elevations for each plane of elevation were performed. In addition to the active arm elevations, measurements were performed during maximal passive internal and external rotation of the upper arm in 90 degrees of elevation in the frontal and sagittal planes, respectively. For these evaluations, the arm of the subject was moved and positioned by the experimenter, with the subject's elbow in about 90 degrees of flexion. The latter measurements were performed for both the ipsilateral and contralateral shoulder of the patients. Even though both the right and left shoulders of the control subjects were evaluated, for this study, only data from the right shoulder were used because differences in observed variables between left and right shoulders were small and not statistically significant.

## Data Processing

Using the reference measurements, for each arm elevation angle, the three-dimensional positions of the bony landmarks on the thorax, scapula, and humerus were calculated in the global (transmitter) coordinate system. Local coordinate systems were constructed on the reconstructed bony landmark positions<sup>33</sup> (Fig. 1, Table 2). The relative positions of the local coordinate systems were subsequently decomposed and expressed in Euler angles.<sup>16,19,33</sup> Order of decomposition was according to the International Shoulder Group proposal<sup>33</sup> (Table 2). Rotations of scapula and humerus were expressed in the coordinate system of the thorax. The glenohumeral rotations were obtained by calculating the relative position of the humerus with respect to the coordinate system of the scapula. Five rotations were regarded to be of clinical rele-



**FIGURE 1** Local coordinate systems as constructed on the reconstructed bony landmark positions.

vance: the scapular rotation around the (vertical) y-axis of the thorax (i.e., the scapular protraction [external rotation]), the scapular rotation around the (anteroposterior) z- axis of the thorax (i.e., the scapular lateral rotation [upward rotation]), the scapular rotation around the (mediolateral) x- axis of the thorax (i.e., the spinal tilt), the humeral rotation around the y-axis of the humerus (i.e., the axial rotation), and the humeral rotation around the x- axis of the scapula (i.e., the glenohumeral axial rotation).

Maximal thoracohumeral elevation angles were compared using a one-way analysis of variance. To standardize the humeral elevation angles among subjects, data were averaged and smoothed by fitting spline functions through the raw data of the three consecutive trials.<sup>35</sup> The obtained spline functions were subsequently sampled at 30, 50, 70, 90, 110, 120, and 130 degrees of humeral elevation. These smoothed and sampled data were used for further processing. Statistical analyses were only performed at 50, 70, 90, and 110 degrees of humeral elevation because of missing data points. A general linear model multivariate analysis of variance with repeated measures, using humeral elevation as a within-subjects factor, was applied to compare the

measures of humeral axial rotation, scapular lateral rotation, scapular protraction, and scapular spinal tilt of shoulders of the control subjects with the ipsilateral shoulders of the patients with stroke.

The data of the internal/external rotation experiments were processed as single observations, and no smoothing and elevation angle correction was applied. A one-way analysis of variance was applied to compare humeral axial rotation, glenohumeral axial rotation, scapular lateral rotation, and scapular spinal tilt between the shoulders of the control subjects and both the ipsilateral and contralateral shoulders of the patients with stroke. When a significant difference was found, a Scheffé post hoc test was used to determine which groups were different. For all statistical tests, results were considered significant at  $P$  values of  $<0.05$ .

As no previous data existed on the variability of kinematics of stroke patients, we calculated a required sample size based on a normal population.<sup>18</sup> In this study, the interindividual variability of scapular rotations was found to be about 7 degrees. With an alpha of 0.05 and a desired smallest detectable difference of 10 degrees, a sample size of  $n = 10$  resulted in a power of 0.8.

## RESULTS

### General Remarks

An example of the raw data with the fitted spline is presented in Figure 2. Each data point in this graph represents one observation (i.e., one measurement of the relative position of a bone with respect to one axis of the thorax at one humeral elevation angle). This particular example represents the rotation of the scapula around the z-axis of the thorax (i.e., the scapular lateral rotation). Graphic representations of the combined results are presented in Figures 3 and 4. In Figure 3, for each elevation plane, the Euler angles of the three scapular rotations and the humeral axial rotation have been plotted as a function of humeral elevation. Box-plot format, indicating median (*horizontal line*), 50% of observations (*box*), and range (*whiskers*), was used. In Figure 4, the same was performed for the internal/external rotation experiments.

### Arm Elevations

Maximal active thoracohumeral elevation angles were significantly ( $P < 0.01$ ) lower in patients ( $125.7 \pm 11.6$  degrees) than in the control subjects ( $137.7 \pm 8.0$  degrees). Comparison of the nonparetic, ipsilateral shoulder of the patients to the shoulders of the control group revealed a significant group-angle interaction for scapular protraction during elevation in the sagittal plane, indicating that the increase in protraction with increasing humeral elevation was higher in control



**TABLE 2** Definitions of bony landmarks: coordinate systems and Euler angle decomposition/rotation order per bone

| Bone    | Bony Landmarks  | Coordinate Systems   | Rotation Order |
|---------|---|--|----------------|
| Thorax  | PX, processus xiphoideus<br>IJ, incisura jugularis<br>C7, processus spinosi C7<br>T8, processus spinosi T8<br>Origin, IJ      | y-axis: line connecting midpoint PX-T8 and midpoint IJ-C7, pointing upwards; x-axis: line perpendicular to plane IJ, C7, and midpoint PX-T8, pointing laterally to the right; z-axis perpendicular to y- and z-axis, pointing backward | X, Y', Z''     |
| Scapula | AA, angulus acromialis<br>TS, trigonum spinae<br>AI, angulus inferior<br>Origin, AA   | x-axis: line connecting TS and AA, pointing laterally to the right; z-axis: perpendicular to the scapular plane, pointing backward; y-axis: line perpendicular to x- and z-axis, pointing upward                                       | Y, Z', X''     |
| Humerus | EL, epicondylus lateralis<br>EM, epicondylus medialis<br>GH, glenohumeral joint<br>rotation center<br>Origin, GH <sup>a</sup> | y-axis: line connecting midpoint EL-EM and GH, pointing upward; z-axis: perpendicular to plane formed by y-axis and line connecting EL-EM, point backward; x-axis: perpendicular to y- and z-axis, pointing laterally to the right     | Y, Z', Y''     |

<sup>a</sup> Not a true bony landmark but estimated from regression equations (Meskers et al. 1997).

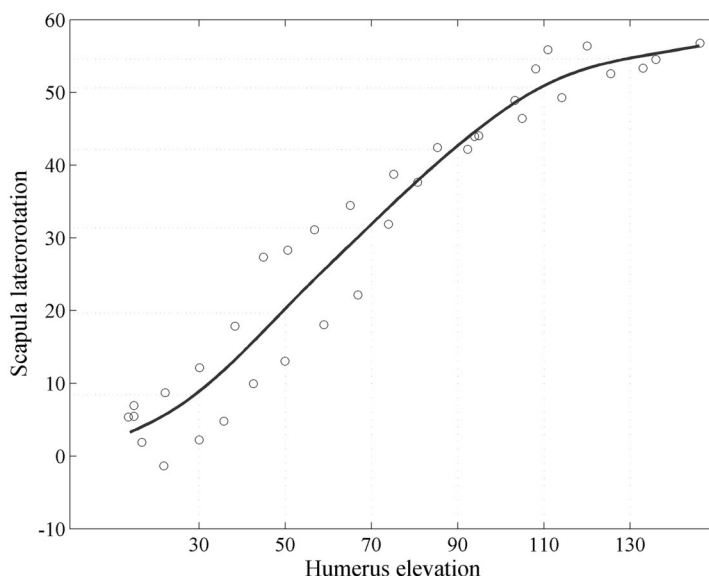
subjects. Scapular protraction during humeral elevation in the sagittal plane was found to be statistically diminished ( $38 \pm 5$  vs.  $51 \pm 8$  degrees), and there was significantly less humeral external rotation during elevation in the frontal plane ( $51 \pm 7$  vs.  $69 \pm 14$  degrees) (Fig. 3).

### Internal/External Rotations

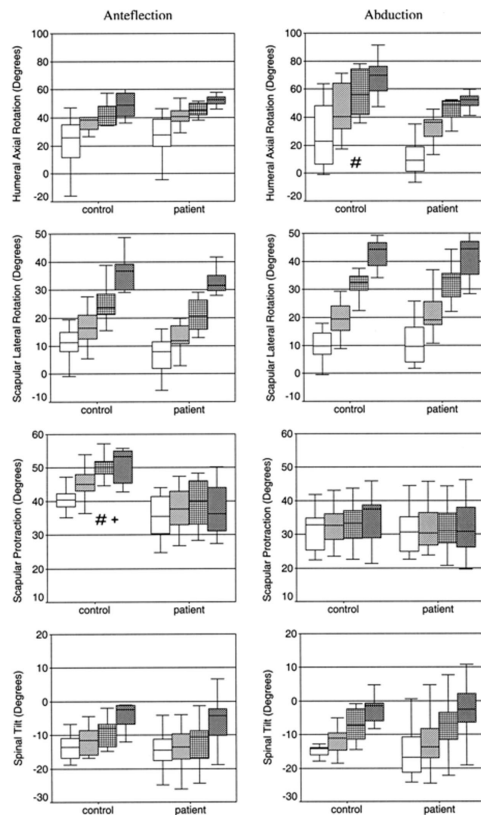
In the internal/external rotation experiments, a statistically significant lower maximal passive external rotation of the humerus in the sagittal plane was found ( $65 \pm 11$  vs.  $94 \pm 12$  degrees). Both

maximal passive internal and external rotation were significantly impaired during passive axial rotation in the frontal plane ( $-19 \pm 19$  vs.  $-49 \pm 20$  degrees and  $64 \pm 13$  vs.  $98 \pm 14$  degrees).

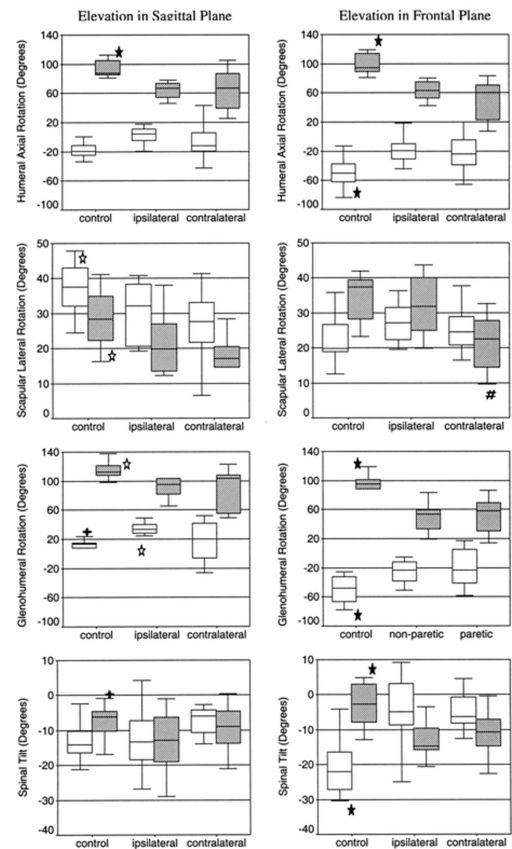
Glenohumeral axial rotation was significantly impaired during internal rotation in the sagittal plane ( $11 \pm 13$  vs.  $38 \pm 16$  degrees) and during both internal and external rotation in the frontal plane ( $-26 \pm 17$  vs.  $-49 \pm 20$  degrees and  $51 \pm 20$  vs.  $96 \pm 20$  degrees). Scapular posterior spinal tilt was significantly diminished during external rotation in the sagittal plane ( $-7 \pm 6$  vs.  $-14 \pm 9$



**FIGURE 2** An example of the raw data with the fitted P-spline. Each data point in this graph represents one observation (i.e., one measurement of the relative position of a bone with respect to one axis of the thorax at one humeral elevation angle). This example represents the rotation of the scapula around the z-axis of the thorax (i.e., the scapular lateral rotation).



**FIGURE 3** Results from the arm elevation experiments. The figures show Euler angles of the humeral axial rotation and the three scapular rotations plotted as a function of humeral elevation angle (from left to right: 50, 70, 90, and 110 degrees) averaged for the control group and the patient group (ipsilateral shoulder). The left figures contain data from the movement in the sagittal plane (anteflexion), and the right figures contain data from the movement in the frontal plane (abduction). The box plot format indicates median (horizontal line), 50% of observations (box), and range (whiskers). #, significantly different; +, significant interaction.



**FIGURE 4** Results from the maximal passive internal (open bars) and external rotation (dashed bars) tests of the humerus in 90 degrees of elevation in the sagittal (anteflexion, left figures) and frontal (abduction, right figures) plane. The figures show Euler angles of the humeral and glenohumeral axial rotation, the scapula lateral rotation, and the scapular spinal tilt averaged for the control group and the patient group (both ipsilateral nonparetic and contralateral paretic shoulders). Solid star, different from nonparetic and paretic; open star, different from paretic; #, different from control and nonparetic; +, different from nonparetic.

degrees) and frontal plane ( $-3 \pm 6$  vs.  $-14 \pm 8$  degrees) and enhanced during internal rotation in the frontal plane ( $-20 \pm 9$  vs.  $-5 \pm 10$  degrees). In general, the patterns of the ipsilateral shoulder resembled more the paretic shoulder than the shoulder of the controls. Scapular lateral rotation in the ipsilateral shoulder was not statistically different from the control group, although there was a tendency toward lower lateral rotation.

## DISCUSSION

### General Remarks

In short, we found distinct changes in ipsilateral shoulder kinematics, predominantly in the form of

significantly diminished humeral external rotation during arm elevation in the frontal plane and during passive axial rotation in both sagittal and frontal planes and in the form of lags in scapular movement: diminished scapular protraction during elevation in the sagittal plane and diminished posterior spinal tilt during maximal passive external rotation, together with diminished anterior tilt during internal rotation in the frontal plane. Furthermore, there was a tendency toward diminished scapular lateral rotation both during elevation and maximal passive axial rotation in the sagittal plane. There was diminished ( $-8.7\%$ ) maximal thoracohumeral elevation in patients ( $125.7$  vs.  $137.7$  degrees).

It should be noted that the Euler angles of the three rotations are not independent of each other, as they represent one three-dimensional movement. This means that interpretations of isolated rotations should be handled with care and that it is important to keep the total movement pattern in mind. The clinical data on, for example, shoulder pain, muscle tone, and arm functionality were only used to get an overview of the characteristics of the population of stroke patients measured, and because of the small size of the population, no further attempt was made to correlate clinical data to the measurements.

### Possible Causes of the Observed Changes

Several possible causes for the observed kinematic changes in the ipsilateral shoulder emerge from the literature. First, in the acute phase of stroke, cerebral edema seems to be spreading throughout the brain, up into the contralateral hemisphere.<sup>24</sup> This would implicate an impairment of the function of the contralateral hemisphere in stroke as well, which could lead to impairment in strength and coordination in the ipsilateral limb.

Second, several studies have elaborated on the concept of both hemispheres controlling both sides of the body by corticospinal neural pathways that do not cross at the brainstem level. Urban et al.<sup>36</sup> found the respiratory musculature to be responding to stimuli applied to both hemispheres, though the response to stimuli applied at the ipsilateral side was weaker. This indicates that when a hemisphere is damaged due to stroke, corticospinal tracts in the ipsilateral side would be affected as well, though to a lesser extent than the contralateral side. Esparza et al.<sup>27</sup> found evidence that temporal coordination of the upper limb recruitment is mediated bilaterally by each hemisphere, with the left hemisphere to be more important. Sugarman et al.<sup>26</sup> found increased segmentation in the movements of the ipsilateral and the contralateral side of patients with hemiplegia. They assumed a global inability to control motion to be responsible for these findings. The fact that the kinematics of the ipsilateral side more or less resembled kinematics of the paretic side may underline a kind of parallel coordination of both sides of the body.

Third, there is the concept of crowding, meaning that as a result of plasticity of the brain, a number of tasks are taken over by the contralateral side, causing function impairments of the other tasks of the hemisphere as a trade-off.<sup>37–40</sup>

Fourth, it is not known to what extent the kinematics of the ipsilateral shoulder are affected by changes in muscular tone at the affected side of the body. We expressed both humeral and scapular rotations in the coordinate system of the thorax,

reducing the influence of pure thorax rotations. Viewed dynamically, it is theoretically possible that changes in the contralateral side affect dynamics in the ipsilateral side; however, left and right shoulders are a less-coupled system than the hip joints within the pelvic system, where a closed kinematic chain makes rotations in both hip joints influence each other. Hence, it seems less likely that paretic muscles on the other side influence a relatively fixed variable as the scapulohumeral rhythm. To shed more light on the possible relationship between muscle tone on the contralateral side and kinematics on the ipsilateral side, data of a group of patients with clear high tone should be compared with patients with clear low tone. A more indicative study design would be repetitive measurements in the follow-up after stroke to assess kinematics and muscle tone simultaneously. However, if a relationship were found between muscle tone on the contralateral side and kinematics on the ipsilateral side, it would still remain unclear whether this was caused by influences from the contralateral to the ipsilateral side or by direct changes in muscle tone on the ipsilateral side.

Finally, other external causes cannot be ruled out by this study. We chose an age-comparable control group to correct for the possible confounding effect of age, but we did not regard factors such as prolonged inactivity. One way to correct for these influences would have been to measure kinematics in the acute phase of stroke. If kinematic changes were present in the acute phase, then the first two explanations would seem to be the most plausible ones. The finding that the kinematic patterns of the ipsilateral shoulder resembled more the patterns of the paretic shoulder than the control shoulder also supports the concept of impaired cerebral control as the cause of the observed alterations.

The data do not point to capsulitis adhesiva as a factor involved, for this would mean diminished rotations at the glenohumeral joint, causing the rotations of the scapula to be enhanced. The lateral rotation, for example, would show a steeper slope, showing a “scapular lead.”<sup>41</sup> Especially, the diminished scapular spinal tilt during maximal passive internal rotation and persistent spinal tilt during maximal passive external rotation of the upper arm is in contradiction with a diminished glenohumeral mobility.

One may speculate about the persistence of the observed changes. Jung et al.<sup>28</sup> found the lack of strength in the ipsilateral side after stroke to be lasting. Elaboration of the relationship between time after stroke and the degree of kinematic changes was not a goal of the present study, and the number of patients was too small. We are cur-



rently undertaking a study to measure kinematics during the first months after the onset of stroke. An interesting question will be whether the observed changes are only present during a part of the rehabilitation process, implicating a "critical window."

### **Passive vs. Active Motions**

The main difference between passive and active motions is the additive generation of an external moment in the latter case above muscle activation needed for stabilization and integrity of the shoulder in the former. Passive internal and external rotations were included to test the shoulder to its limits by assessing kinematics during maximal humeral rotations. We thus expected to detect aberrations more easily. Substantial differences were indeed found during the passive test, underlining this premise. We argue that differences in kinematics found during passive motions point to the same as differences found during active motions, namely, the existence of an aberrant muscular contraction pattern.

### **Clinical Implications**

The clinical implications of the observed kinematic changes in the present study remain speculative and require further elaboration. It may be that problems in the ipsilateral shoulder in hemiplegia due to stroke cannot be accounted to overuse only, as is commonly done in clinical practice. This is because we found evidence for underlying, potentially harmful, kinematic alterations in the form of inadequate scapular positioning with respect to the humerus, possibly resulting in narrowing of the subacromial space by the major tubercle or overstretch of the scapulohumeral soft tissue. To our knowledge, no information exists on the prevalence of shoulder problems on the ipsilateral side in stroke patients, nor is it clear to what extent problems of the ipsilateral shoulder hamper the rehabilitation process. However, it seems obvious that this is valuable information because stroke patients have to rely heavily on the ipsilateral limb. Shoulder problems can be defined in terms of both functional impairment and occurrence of shoulder pain. Regarding the functional impairment, we found that the patients were still able to use their ipsilateral shoulder, though the maximal thoracohumeral elevation angle was less than in the control group. Jung et al.<sup>28</sup> found muscle strength to be impaired, which may be a likely explanation of this lower maximal elevation angle. Price et al.,<sup>41</sup> studying scapular positions in the hemiplegic shoulder of stroke patients, found a scapular lag (i.e., a diminished scapular lateral rotation that was positively correlated with the impaired function). A tendency for scapular lag was also found in the

present study. How a scapular lag should be interpreted is not fully clear. Lack of muscle strength might result in both diminished elevation and scapular lag. There might also be a causal relationship between scapular lag and shoulder function. Scapular lag may reveal a problem in stabilizing the scapula, a prerequisite for proper arm function. Suboptimal positioning of the scapula with respect to the arm would lead to suboptimal length of muscles and, thus, to impaired muscle function and abnormal muscle forces. Lack of coordination is then the main underlying problem. The diminished protraction and spinal tilt can be discussed in the same way. To distinguish between cause and effect, simulations by three-dimensional musculoskeletal models of the shoulder are required.

Shoulder pain may develop as a result of impingement, which is likely to occur as a result of both inadequate positioning of the scapula and diminished axial rotation of the humerus, causing the major tubercle to be rotated away from the coracoacromial arch ineffectively. Both the diminished protraction during elevation in the sagittal plane and the lower axial rotation of the humerus during elevation in the frontal plane will enhance the danger of the major tubercle narrowing the subacromial space.<sup>42</sup> This means that the ipsilateral shoulder of stroke patients could be at risk for developing impingement-related shoulder problems. None of the patients we measured experienced pain in the ipsilateral side, but the sample size was too small to draw definite conclusions on this subject, and longer follow-up is needed for further evaluation.

### **CONCLUSION**

We found evidence for improper biomechanical function of the ipsilateral shoulder in stroke (i.e., improper stabilization of the scapula and a diminished ability for external rotation of the upper arm). The clinical consequences of the observed kinematic alterations are not clear yet. Meanwhile, both researchers and clinicians should realize that normal function of the ipsilateral shoulder in stroke cannot be taken for granted. For researchers, this is important in kinematic shoulder research concerning the paretic shoulder, in which kinematic and dynamic models may help to understand poststroke shoulder pain and may help to plan treatment modalities in restoring shoulder function. In kinematic studies, the ipsilateral shoulder should not be automatically used as a control. For the clinician, special care may be necessary to keep the ipsilateral shoulder in stroke patients in optimal shape. This is important because proper function of the ipsilateral arm in stroke is crucial for maintaining self-support.

## REFERENCES

- Broeks JG, Lankhorst GJ, Rumping K, et al: The long-term outcome of arm function after stroke: Results of a follow-up study. *Disabil Rehabil* 1999;21:357-64
- Nakayama H, Jorgensen HS, Raaschou HO, et al: Recovery of upper extremity function in stroke patients: The Copenhagen Stroke Study. *Arch Phys Med Rehabil* 1994;75:394-8
- Jespersen HF, Jorgensen HS, Nakayama H, et al: Shoulder pain after a stroke. *Int J Rehabil Res* 1995;18:273-6
- Poulin de Courval L, Barsauskas A, Berenbaum B, et al: Painful shoulder in the hemiplegic and unilateral neglect. *Arch Phys Med Rehabil* 1990;71:673-6
- Wanklyn P, Forster A, Young J: Hemiplegic shoulder pain (HSP): Natural history and investigation of associated features. *Disabil Rehabil* 1996;18:497-501
- Roy CW, Sands MR, Hill LD: Shoulder pain in acutely admitted hemiplegics. *Clin Rehabil* 1994;8:334-40
- Hakuno A, Sashika H, Ohkawa T, et al: Arthrographic findings in hemiplegic shoulders. *Arch Phys Med Rehabil* 1984;65:706-11
- Najenson T, Yacubovich E, Pikielni SS: Rotator cuff injury in shoulder joints of hemiplegic patients. *Scand J Rehabil Med* 1971;3:131-7
- Gamble GE, Barberan E, Laasch HU, et al: Poststroke shoulder pain: A prospective study of the association and risk factors in 152 patients from a consecutive-cohort of 205 patients presenting with stroke. *Eur J Pain* 2002;6:467-74
- Snels IAK: *Stroke and Shoulder Pain: The Effect of Triamcinolone Acetonide Injections* (dissertation). Amsterdam, Vrije Universiteit, 2001
- Hogfors C, Sigtholm G, Herberts P: Biomechanical model of the human shoulder: I. Elements. *J Biomech* 1987;20:157-66
- Hogfors C, Sigtholm G, Herberts P: Biomechanical model of the human shoulder joint: II. The shoulder rhythm. *J Biomech* 1991;24:699-709
- Veeger HEJ, Van der Helm FCT, Van der Woude LHV, et al: Inertia and muscle contraction parameters for musculoskeletal modelling of the shoulder mechanism. *J Biomech* 1991;24:615-29
- van der Helm FCT: A finite element musculoskeletal model of the shoulder mechanism. *J Biomech* 1994;27:551-69
- van der Helm FCT: Analysis of the kinematic and dynamic behavior of the shoulder mechanism. *J Biomech* 1994;27:527-50
- van der Helm FCT, Pronk GM: Three-dimensional recording and description of motions of the shoulder mechanism. *J Biomech Eng* 1995;117:27-40
- van der Helm FCT, Veeger HEJ: Quasi-static analysis of muscle forces in the shoulder mechanism during wheel-chair propulsion. *J Biomech* 1996;29:39-52
- Meskers CGM, Vermeulen HM, de Groot JH, et al: 3D shoulder position measurements using a six-degree-of-freedom electromagnetic tracking device. *Clin Biomech* 1998;13:280-92
- Karduna AR, McClure PW, Michener LA: Scapular kinematics: Effects of altering the Euler angle sequence of rotations. *J Biomech* 2000;33:1063-8
- de Groot JH: The variability of shoulder motions recorded by means of palpation. *Clin Biomech* 1997;12:461-72
- Johnson GR, Stuart PR, Mitchell S: A method for the measurement of three dimensional scapular movement. *Clin Biomech* 1993;8:269-73
- Sbarbati A, Reggiani A, Nicolato E, et al: Regional changes in the contralateral "healthy" hemisphere after ischemic lesions evaluated by quantitative T2 parametric maps. *Anat Rec* 2002;266:118-22
- Leff A, Crinion J, Scott S, et al: A physiological change in the homotopic cortex following left posterior temporal lobe infarction. *Ann Neurol* 2002;51:553-8
- Izumi Y, Haida M, Hata T, et al: Distribution of brain oedema in the contralateral hemisphere after cerebral infarction: Repeated MRI measurement in the rat. *J Clin Neurosci* 2002;9:289-93
- Pohl PS, Luchies CW, Stoker-Yates J, et al: Upper extremity control in adults post stroke with mild residual impairment. *Neurorehabil Neural Repair* 2000;14:33-41
- Sugarman H, Avni A, Nathan R, et al: Movement in the ipsilesional hand is segmented following unilateral brain damage. *Brain Cogn* 2002;48:579-87
- Esparza DY, Archambault PS, Winstein CJ, et al: Hemispheric specialization in the co-ordination of arm and trunk movements during pointing in patients with unilateral brain damage. *Exp Brain Res* 2003;148:488-97
- Jung HY, Yoon JS, Park BS: Recovery of proximal and distal arm weakness in the ipsilateral upper limb after stroke. *NeuroRehabilitation* 2002;17:153-9
- Lee KC, Carson L, Kinnin E, et al: The Ashworth Scale: A reliable and reproducible method of measuring spasticity. *J Neurorehabil* 1989;3:205-9
- Fugl-Meyer AR, Jaasko L, Leyman I, et al: The post-stroke hemiplegic patient: I. A method for evaluation of physical performance. *Scand J Rehabil Med* 1975;7:13-31
- Berglund K, Fugl-Meyer AR: Upper extremity function in hemiplegia: A cross-validation study of two assessment methods. *Scand J Rehabil Med* 1986;18:155-7
- Meskers CGM, Fraterman H, van der Helm FCT, et al: Calibration of the "Flock of Birds" electromagnetic tracking device and its application in shoulder motion studies. *J Biomech* 1999;32:629-33
- Van der Helm FCT: *A Standardized Protocol for the Description of Shoulder Motion Studies: First Proposal*. Available at: <http://www.internationalshouldergroup.org>. Accessed November 23, 2004
- Meskers CGM, van der Helm FCT, Rozendaal LA, et al: In vivo estimation of the glenohumeral joint rotation center from scapular bony landmarks by linear regression. *J Biomech* 1997;31:93-6
- Eilers PHC, Marx BD: Flexible smoothing with B-splines and penalties. *Stat Sci* 1996;11:89-121
- Urban PP, Morgenstern M, Brause K, et al: Distribution and course of cortico-respiratory projections for voluntary activation in man: A transcranial magnetic stimulation study in healthy subjects and patients with cerebral ischemia. *J Neurol* 2002;249:735-44
- Nass R, Sadler AE, Sidtis JJ: Differential effects of congenital versus acquired unilateral brain injury on dichotic listening performance: Evidence for sparing and asymmetric crowding. *Neurology* 1992;42:1960-5
- Nass R, DeCoudres Peterson H, Koch D: Differential effects of congenital left and right brain injury on intelligence. *Brain Cogn* 1989;9:258-66
- Saykin AJ, Gur RC, Sussman NM, et al: Memory deficits before and after temporal lobectomy: Effect of laterality and age of onset. *Brain Cogn* 1989;9:191-200
- Strauss E, Satz P, Wada J: An examination of the crowding hypothesis in epileptic patients who have undergone the carotid amytal test. *Neuropsychologia* 1990;28:1221-7
- Price CI, Franklin P, Rodgers H, et al: Non-invasive evaluation of shoulder problems after stroke. *Lancet* 1999;353:298
- Meskers CGM, van der Helm FCT, Rozing PM: The size of the supraspinatus outlet during elevation of the arm in the frontal and sagittal plane: A 3-D model study. *Clin Biomech* 2002;17:257-66